DISPERSAL, DISEASE AND LIFE HISTORY EVOLUTION

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Abstract: Single patch discrete- (S-I-S) models are capable of supporting multiple endemic equilibria coexisting with a locally asymptotically stable disease-free equilibrium, via backward bifurcations. We illustrate the richness generated by such "simple" nonlinear systems in the study of two patch epidemic models with disease-enhanced or disease-suppressed dispersal. It is shown that disease persistence can be enhanced by dispersal.

Dispersal, Disease and Life-History Evolution

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Abstract: Single patch discrete-time *S-I-S* epidemic models are capable of supporting multiple endemic equilibria coexisting with a locally asymptotically stable disease-free equilibrium, via backward bifurcations. We illustrate the richness generated by such "simple" nonlinear systems in the study of two patch epidemic models with disease-enhanced or disease-suppressed dispersal. It is shown that disease persistence can be enhanced by dispersal.

1. Introduction

Over the last twenty years (with many notable exceptions) the study of disease dynamics has focused on models with human hosts (see [3, 5, 8, 9, 12, 20, 21, 27]). The impact of "disease" on animal populations in some sense is part of the study of host-parasite interactions (see [20, 25, 26]). Models that incorporate population and disease dynamics as well as dispersal are rare. We study the role of dispersal on disease dynamics within populations capable of supporting complex dynamics. We model local disease dynamics via a susceptible-infective-susceptible (S-I-S) epidemic process. This is clearly unrealistic as most diseases in animal populations are not of this type. We also assume no disease-induce mortality, again, a terribly simplistic assumption.

Our choice of framework is a function of our driving questions: Do complex population dynamics drive disease dynamics? Does dispersal play a key role on disease dynamics? Our setting assumes that complexity comes exclusively from the (disease-free) population dynamics. Hence, it is possible to focus exclusively on the role of dispersal on the dynamics of the infectious subpopulations.

Single patch discrete-time susceptible-infective-susceptible (S-I-S) epidemic models are capable of generating complex (chaotic) dynamics, a situation not shared by classical continuous-time epidemic models [10, 11, 14, 15]. Typically, the reproductive number of the disease is the key threshold parameter. Typically, an R_0 less than one implies the global asymptotic stability of the disease-free equilibrium (the disease dies out), while an R_0 bigger than one typically supports the existence and global asymptotic stability of a unique endemic equilibrium (Theorem 2.1, Corollary 2.2 and Theorem 2.3).

Non-constant transmission rates can generate multiple stable equilibria. Examples of epidemic models exhibiting this behavior were first established for continuous-time epidemic models by Castillo-Chavez *et al.* [8, 9] and Huang *et al.* [23]. Recently, examples using simpler continuous-time models were constructed by Castillo-Chavez and Hadeler [19], Feng *et al.* [17], Kribs *et al.* [24] and P. van den Driessche *et al.*[29]. The results of Feng *et al.* [17], Huang *et al.* [23] and Castillo-Chavez and Hadeler [19] have far reaching implications for the implementation of effective public health policies for HIV and Tuberculosis. The results of P. van den Driessche and P. Watmough have important theoretical implications since they illustrate the possibility of backward bifurcations for simple epidemic processes (S-I-S epidemic models based on a Volterra integral equation [29]). Backward bifurcations are possible in classical discrete-time epidemic models. An example using a discrete-time susceptible-exposed-infective-susceptible (S-E-I-S) epidemic model has been recently developed [4]. Here, we illustrate the possibility of backward bifurcations in simple discrete-time S-I-S epidemic models with or without dispersion between patches.

The paper is organized as follows: In Section 2, we study single patch discrete-time S-I-S models and establish conditions for the occurrence of a unique stable endemic equilibrium point under various recruitment regimes of new susceptibles. In Section 3, we illustrate the possibility of multiple endemic equilibria via a backward bifurcation in single patch discrete-time S-I-S models. Two patch discrete-time S-I-S models with dispersion between patches are introduced in Section 4. Section 5 focuses on the role of disease-enhanced and disease suppressed dispersal on disease persistence. In Section 5, it is also shown that disease persistence with dispersal between patches is possible in situations where the disease

would be on the brink of extinction in the absence of dispersal. Conclusions are in Section 6 and proofs are collected in Section 7, the Appendix.

2. S-I-S Epidemic Models

The dynamics of the total population size in generation t and Patch $i \in \{1, 2\}$, denoted by $T_i(t)$, is governed by the equation $T_i(t+1) = f_i(T_i(t)) + \gamma_i T_i(t)$ whenever there is no dispersion. Here, γ_i denotes the probability of survivorship per generation while f_i denotes the local birth or recruitment functions. In general, f_i is a nonlinear function capable of generating complex dynamics. An epidemic process is built on "top" of the demographic pattern generated by f_i, in each patch. To guarantee control over the local dynamics (no matter how complex they are) it is assumed that the disease does not affect T_i in a *significant* way. Fixing the local population dynamics and building an epidemic processes on them is quite common for continuous-time epidemic processes but less common for discrete-time epidemic models [1, 2, 10, 11].

$S_i(t)$ denotes the population of susceptibles; $I_i(t)$ denotes the population of infecteds, assumed infectious; $T_i(t) \equiv S_i(t) + I_i(t)$ denotes the total population size at generation t, and, $T_{i\infty} \equiv \lim_{t\to\infty} T_i(t)$ denotes the demographic steady state for the total population whenever it exists. Individuals are assumed to survive with constant probability γ_i (die with probability $1-\gamma_i$) each generation while infected individuals recover with probability $1-\sigma_i$ (do not recover with constant probability σ_i). It is assumed that susceptible individuals become infected with probability $1 - G_i$ per generation (remain susceptible with non-constant probability function G_i), where $G_i \equiv G_i(\alpha_i(\frac{I_i(t)}{T_i(t)}))$ and $\alpha_i \equiv \alpha_i(\frac{I_i(t)}{T_i(t)})$. That is, α_i models the impact of

prevalence ($\frac{I_i(t)}{T_i(t)}$) on $G_i(\alpha_i = \frac{I_i(t)}{T_i(t)}$ implies $\alpha_i \leq 1)$. In general, $G_i : [0, \infty) \to [0, 1]$ is a monotone function with $G_i(0) = 1; G_i'(x) < 0$ and $G_i''(x) \geq 0$ for all $x \in [0, \infty)$.

It is also assumed that the disease is not fatal; all recruits are susceptible; the recruitment function depends on the total population; time is measured in generations; and, recovery from disease does not give permanent or temporary immunity. Model construction assumes implicitly a sequential process. At each generation, the fraction $(1 - \gamma_i)$ of each class is removed (death); surviving susceptibles then become infected with probability $(1 - G_i)$; while, independently, surviving infectives recover with probability $(1 - \sigma_i)$, in Patch $i \in \{1, 2\}$. The use of a sequential approach simplifies the analysis without limiting the nature of the results.

Our assumptions lead to the following single patch, discrete-time $S - I - S$ model with no dispersion between patches:

$$\left.\begin{array}{rcl} S_i(t+1) & = & f_i(T_i(t)) + \gamma_i G_i(\alpha_i(\frac{I_i(t)}{T_i(t)})\frac{I_i(t)}{T_i(t)})S_i(t) + \gamma_i(1-\sigma_i)I_i(t), \\ I_i(t+1) & = & \gamma_i(1 - G_i(\alpha_i(\frac{I_i(t)}{T_i(t)})\frac{I_i(t)}{T_i(t)}))S_i(t) + \gamma_i\sigma_i I_i(t), \end{array}\right\} \quad (1)$$

where $0 < \gamma_i, \sigma_i < 1$, $T_i(t) > 0$, and $\alpha_i(\frac{I_i(t)}{T_i(t)}) \geq 0$. Model (1) reduces to the model of Castillo-Chavez and Yakubu whenever the transmission function $\alpha_i(\frac{I_i(t)}{T_i(t)})$ is a constant [6, 10].

The population is regulated by density and not by disease. Hence, the absence of disease-induced mortality does not imply that the population does not experience death. Density regulation can support complex dynamics for $T_i(t)$. Whether or not they are qualitatively identical to the disease dynamics is part of the questions of interest (see [6, 10]). From System (1), we have that

$$T_i(t+1) = f_i(T_i(t)) + \gamma_i T_i(t) \qquad \text{for } i \in \{1, 2\}, \qquad (2)$$

that is, the total population dynamics depends exclusively on births (recruits) and deaths. Equation (2) describes the local population dynamics in each patch in the absence of dispersion.

2.1. Asymptotically Bounded Growth

To gain some understanding on the role of dispersal, population dynamics, and disease on life-history evolution, we look at the dynamics of our model under specific functional forms for the recruitment function f_i (forms commonly found in the literature). If we assume that new recruits arrive on Patch i at the positive constant rate Λ_i per generation and no dispersion, Equation (2) with $f_i(T_i(t)) = \Lambda_i$ implies that the total population will eventually reach the positive study state $T_{i\infty} = \frac{\Lambda_i}{1-\gamma_i}$. If the birth or recruitment process is governed by Ricker's Equation, $f_i(T_i(t)) = T_i(t)\exp(r_i - k_i T_i(t))$ where r_i and k_i are positive constants, then Equation (2) implies that the total population, on each patch, will eventually reach a positive steady state

$$T_{i\infty} = \frac{r_i - \ln(1-\gamma_i)}{k_i}$$

provided that

$$0 < r_i < \frac{2 + (1-\gamma_i)\ln(1-\gamma_i)}{(1-\gamma_i)}$$

($T_i(t)$ would exhibit complex local dynamics for larger values of r_i[see, 10]).

Hence, we assume that the total population ($T_i(t) \equiv S_i(t) + I_i(t)$) on Patch i has reached the positive steady state $T_{i\infty}$ and, set $S_i(t) = T_{i\infty} - I_i(t)$. The resulting one-dimensional autonomous "limiting system" for $I_i(t)$ is defined by this substitution. The single-patch limiting equation for the local dynamics in

Patch i is given by

$$I_i(t+1) = \gamma_i(1 - G_i(\alpha_i(\tfrac{I_i(t)}{T_{i\infty}})\tfrac{I_i(t)}{T_{i\infty}}))(T_{i\infty} - I_i(t)) + \gamma_i\sigma_i I_i(t). \qquad (3)$$

Simulations support the conclusion that Equation (3) exhibits the same qualitative dynamics as those of System (1). Theoretical results on the qualitative dynamics equivalence of autonomous and non-autonomous systems have been established by Thieme [28] but in the context of continuous-time dynamical systems. The following results depend on the assumption that Equation (3) and System (1) have the same qualitative dynamics near a stable equilibrium population size.

The local patch basic reproductive number, $\Re_{0i}$, determines the uncoupled (no dispersion) asymptotic behavior of System (3).

$$\Re_{0i} = \begin{cases} \gamma_i\sigma_i & if \quad \alpha_i(0) = 0, \\ \frac{-\gamma_i\alpha_i(0)G_i'(0)}{1-\gamma_i\sigma_i} & if \quad \alpha_i(0) \neq 0, \end{cases} \qquad (4)$$

gives the average number of secondary infections generated by a small pioneer population of infected (assumed infectious) individuals over their life-time in Patch i (in the absence of dispersion). Epidemiologically (and typically), if $\Re_{0i} > 1$ the number of infectives in Patch i (no dispersion) grows while if $\Re_{0i} \leq 1$ the number of infectives would decrease to zero regardless of initial conditions. We collect these results in the following theorem:

Theorem 2.1. *Let*

$$\alpha_i(\frac{I_i}{T_{i\infty}}) + \frac{I_i}{T_{i\infty}}\frac{d\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i} > 0$$

and

$$2\frac{d\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i} + \frac{I_i}{T_{i\infty}}\frac{d^2\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i^2} \leq 0$$

in Patch $i \in \{1, 2\}$.

(a) If $\Re_{0i} \leq 1$, then the solutions $(S_i(t), I_i(t))$ of System (3) approach the disease free equilibrium, $(T_{i\infty}, 0)$, as $t \to \infty$.

(b) If $\Re_{0i} > 1$, then the solutions $(S_i(t), I_i(t))$ of System (3) approach a unique positive endemic equilibrium, $(\overline{S}_i, \overline{I}_i) \in (0, \infty) \times (0, \infty)$, as $t \to \infty$.

The proof of Theorem 2.1 is in the Appendix. The following result is an immediate consequence of Theorem 2.1.

Corollary 2.2. *Let $\alpha_i \equiv \alpha_i(\frac{I_t}{T_{i\infty}})$ be a positive constant in Patch $i \in \{1, 2\}$.*

(a) If $\Re_{0i} \leq 1$, then the solutions $(S_i(t), I_i(t))$ of System (3) approach the disease free equilibrium, $(T_{i\infty}, 0)$, as $t \to \infty$.

(b) If $\Re_{0i} > 1$, then the solutions $(S_i(t), I_i(t))$ of System (3) approach a unique positive endemic equilibrium, $(\overline{S}_i, \overline{I}_i) \in (0, \infty) \times (0, \infty)$, as $t \to \infty$.

2.2. Geometric Growth

If new recruits arrive on Patch i at the positive per-capita rate μ_i per generation, that is, if $f_i(T_i(t)) = \mu_i T_i(t)$ then Equation (2) reduces to the linear difference equation

$$T_i(t+1) = (\mu_i + \gamma_i) T_i(t), \qquad (5)$$

that is,

$$T_i(t) = (\mu_i + \gamma_i)^t T_i(0). \qquad (6)$$

We define the local demographic basic reproductive number (no dispersion) as

$$\Re_{id} = \frac{\mu_i}{1 - \gamma_i}.$$

$\Re_{id}$ is a dimensionless quantity that gives the average number of descendants produced by a small pioneer population ($T_i(0)$) over its life-time in Patch i. Hence, if $\Re_{id} > 1$, the population invades Patch i at a geometric rate while if $\Re_{id} < 1$ the population dies geometrically, in Patch i. We now build an epidemic process on a population with intrinsic geometric dynamics.

The study of the dynamics of System (1) can be simplified via the use of proportions. The use of the new variables

$$x_i(t) = \frac{S_i(t)}{T_i(t)} \text{ and } y_i(t) = \frac{I_i(t)}{T_i(t)}, \qquad (7)$$

reduces System (1) with $f_i(T_i(t)) = \mu_i T_i(t)$ to:

$$\left.\begin{array}{rcl} x_i(t+1) & = & \frac{\mu_i}{\mu_i+\gamma_i} + \frac{\gamma_i}{\mu_i+\gamma_i} x_i(t) G_i(\alpha_i(y_i(t))y_i(t)) + \frac{\gamma_i}{\mu_i+\gamma_i}(1-\sigma_i)y_i(t), \\ y_i(t+1) & = & \frac{\gamma_i}{\mu_i+\gamma_i} x_i(t)(1 - G_i(\alpha_i(y_i(t))y_i(t))) + \frac{\gamma_i}{\mu_i+\gamma_i}\sigma_i y_i(t). \end{array}\right\} \quad (8)$$

System (8) reduces to the single patch model of Castillo-Chavez and Yakubu whenever the transmission function $\alpha_i(\frac{I_i(t)}{T_i(t)})$ is a constant [10]. Since $x_i(t) + y_i(t) = 1$ for all t in System (8) then all solutions live on the invariant line $\{(x_i, y_i) \in [0,\infty) \times [0,\infty) \mid x_i + y_i = 1\}$. The substitution $x_i(t) = 1 - y_i(t)$ reduces System (8) to a one-dimensional autonomous "system" for $y_i(t)$, namely,

$$y_i(t+1) = \frac{\gamma_i}{\mu_i+\gamma_i}(1 - y_i(t))(1 - G_i(\alpha_i(y_i(t))y_i(t))) + \frac{\gamma_i}{\mu_i+\gamma_i}\sigma_i y_i(t). \qquad (9)$$

From Equation (9) we compute the local basic reproductive number (no dispersion):

$$\Re_{0i} = \begin{cases} \frac{\gamma_i\sigma_i}{(1-\Re_{id})\gamma_i+\Re_{id}} & if \quad \alpha_i(0) = 0, \\ \frac{-\gamma_i\alpha_i(0)G_i'(0)}{(1-\gamma_i)(\Re_{id}-1)+1-\gamma_i\sigma_i} & if \quad \alpha_i(0) \neq 0. \end{cases} \qquad (10)$$

$\Re_{0i}$ is easily derived from the linearization of Equation (9) near $(x_{i\infty}, y_{i\infty}) \equiv (1, 0)$, that is, from

$$y_i(t+1) \approx \frac{\gamma_i}{\mu_i+\gamma_i}(-\alpha_i(0)G_i'(0) + \sigma_i)y_i(t).$$

If $\Re_{id} = 1$ (no demographic impact in Patch i) then $\Re_{0i}$ reduces to $\Re_{0i} = \gamma_i \sigma_i$ or $\Re_{0i} = \frac{-\gamma_i \alpha_i(0) G_i'(0)}{1-\gamma_i \sigma_i}$ where $\frac{1}{1-\gamma_i \sigma_i}$ denotes the average death-adjusted length of the infectious period in generations; γ_i is the proportion of surviving susceptibles who can be invaded by the disease; and, $-\alpha_i(0)G_i'(0)$ is the maximum rate of infection per infective in Patch i (no dispersion) (see [10]). If $\Re_{id} \neq 1$ then demography impacts disease dynamics, that is $\Re_{0i}$. In fact, $\frac{1}{(1-\gamma_i)(\Re_{id}-1)+1-\gamma_i \sigma_i}$ gives the demographic death-adjusted infectious period measured in generations. Hence, $\Re_{0i}$ decreases with population growth ($\Re_{id} > 1$) and increases with population decay ($0 < \Re_{id} < 1$) as all new recruits are assumed to be susceptibles. We collect these results in the following theorem:

Theorem 2.3. *In System (8), let* $f_i(T_i) = \mu_i T_i$, $\alpha_i(y_i) + y_i \frac{d\alpha_i(y_i)}{dy_i} > 0$ *and* $2\frac{d\alpha_i(y_i)}{dy_i} + y_i \frac{d^2\alpha_i(y_i)}{dy_i^2} \leq 0$ *in Patch* $i \in \{1, 2\}$. *Then,*

(a) if $\Re_{id} < 1$, *the total population,* $T_i \equiv S_i + I_i$, *decreases to zero at a geometric rate;* $\Re_{id} > 1$ *implies that the total population increases at a geometric rate;* $\Re_{id} = 1$ *implies that the total population remains fixed at its initial value.*

(b) If $\Re_{id} > 1$ *and* $\Re_{0i} \leq 1$, *then the proportion* $\frac{I_i}{T_i}$ *of infectives in the total population tends to* 0 *as* $t \to \infty$, *while the proportion* $\frac{S_i}{T_i}$ *of susceptibles in the total population tends to* 1 *as* $t \to \infty$. *Hence,* $(\frac{S_i}{T_i}, \frac{I_i}{T_i})$ *tends to the disease-free equilibrium* $(1, 0)$, *where* S_i *is increasing at the same geometric rate as* T_i.

(c) If $\Re_{id} > 1$ *and* $\Re_{0i} > 1$, *then the proportion* $\frac{I_i}{T_i}$ *of infectives in the total population tends to a positive number,* $\overline{\frac{I_i}{T_i}}$ *as* $t \to \infty$, *and the proportion* $\frac{S_i}{T_i}$ *of susceptibles in the total population also tends to a positive number* $\overline{\frac{S_i}{T_i}}$ *as* $t \to \infty$. *Hence,* $(\frac{S_i}{T_i}, \frac{I_i}{T_i})$ *tends to an endemic equilibrium.* I_i, S_i *and* T_i *are increasing at the same geometric rate.*

(d) If $\Re_{id} < 1$ *and* $\Re_{0i} \leq 1$, *then the proportion* $\frac{I_i}{T_i}$ *of infectives in the total population tends to* 0 *as* $t \to \infty$, *while the proportion* $\frac{S_i}{T_i}$ *of susceptibles in the total population tends to* 1 *as* $t \to \infty$. *Hence,* $(\frac{S_i}{T_i}, \frac{I_i}{T_i})$ *tends to the disease-free equilibrium* $(1, 0)$. *Hence,* S_i *is decreasing to zero at the same geometric rate as* T_i.

(e) If $\Re_{id} < 1$ *and* $\Re_{0i} > 1$, *then the proportion* $\frac{I_i}{T_i}$ *of infectives in the total population tends to a positive number* $\overline{\frac{I_i}{T_i}}$ *as* $t \to \infty$, *and the proportion* $\frac{S_i}{T_i}$ *of susceptibles in the total population also tends to a positive number* $\overline{\frac{S_i}{T_i}}$ *as* $t \to \infty$. *Hence,* $(\frac{S_i}{T_i}, \frac{I_i}{T_i})$ *tends to an endemic equilibrium. Hence,* I_i, S_i *and* T_i *are decreasing at a geometric rate.*

The proof of Theorem 2.3 is in the Appendix.

3. Forward and Backward Bifurcations

Typically, epidemic models have a unique endemic equilibrium with the reproductive number of the disease serving as a threshold parameter (transcritical bifurcation). If the reproduction number is less than one, the disease dies out and if the reproduction number is bigger than one, the disease persists (Theorem 2.1, Corollary 2.2 and Theorem 2.3). That is, we have a forward transcritical bifurcation. If the transmission rate $\alpha_i \equiv \alpha_i(\frac{I_i}{T_i})$ is a non-constant function then multiple endemic equilibria for System (3) are possible even when the basic reproductive number is less than one (backward bifurcation). We illustrate explicitly this possibility later on.

To build such examples, we assume throughout this section that infections are modeled as Poisson processes and that $\alpha_i(y_i) = y_i$ so that $G_i(\alpha_i(y_i)y_i) = e^{-y_i^2}$

[10]. For simplicity, we use the approximation $e^{-y_i^2} \approx 1 - y_i^2$. This last assumption allows us to make explicit computations. System (8) reduces to the well-posed system:

$$\left.\begin{array}{rcl} x_i(t+1) & = & \frac{\mu_i}{\mu_i+\gamma_i} + \frac{\gamma_i}{\mu_i+\gamma_i} x_i(t)(1-y_i(t)^2) + \frac{\gamma_i}{\mu_i+\gamma_i} y_i(t)(1-\sigma_i), \\ y_i(t+1) & = & \frac{\gamma_i}{\mu_i+\gamma_i} y_i(t)^2 x_i(t) + \frac{\gamma_i}{\mu_i+\gamma_i} \sigma_i y_i(t). \end{array}\right\} \quad (11)$$

Since $2\frac{d\alpha_i(y_i)}{dy_i} + y_i \frac{d^2\alpha_i(y_i)}{dy_i^2} > 0$, neither Theorem 2.1 nor Theorem 2.3 applies. From $\alpha_i(0) = 0$ and Equation (10), we have that $\Re_{0i} = \frac{\gamma_i \sigma_i}{(1-\Re_{id})\gamma_i + \Re_{id}} < 1$ *for all values of the parameters*. Theorem 3.1 collects results on the existence of endemic equilibria in System (11) with $\Re_{0i} < 1$ (backward bifurcation):

Theorem 3.1. *(a) If*

$$0 < \Re_{0i} < 1 - \frac{\gamma_i}{4(\mu_i + \gamma_i)},$$

then the solutions $(\frac{S_i}{T_i}, \frac{I_i}{T_i})$ *of System (11) approach the disease free equilibrium,* $(1, 0)$, *as* $t \to \infty$.

(b) If

$$\Re_{0i} = 1 - \frac{\gamma_i}{4(\mu_i + \gamma_i)},$$

then System (11) has an unstable endemic equilibrium at $(\frac{1}{2}, \frac{1}{2})$ *coexisting with the locally asymptotically stable disease free equilibrium at* $(1, 0)$.

(c) If

$$\Re_{0i} > 1 - \frac{\gamma_i}{4(\mu_i + \gamma_i)},$$

then System (11) has an unstable endemic equilibrium at

$$(\frac{1}{2}(1 + \sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1-\Re_{0i})}), \frac{1}{2}(1 - \sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1-\Re_{0i})}))$$

and a locally asymptotically stable endemic equilibrium at

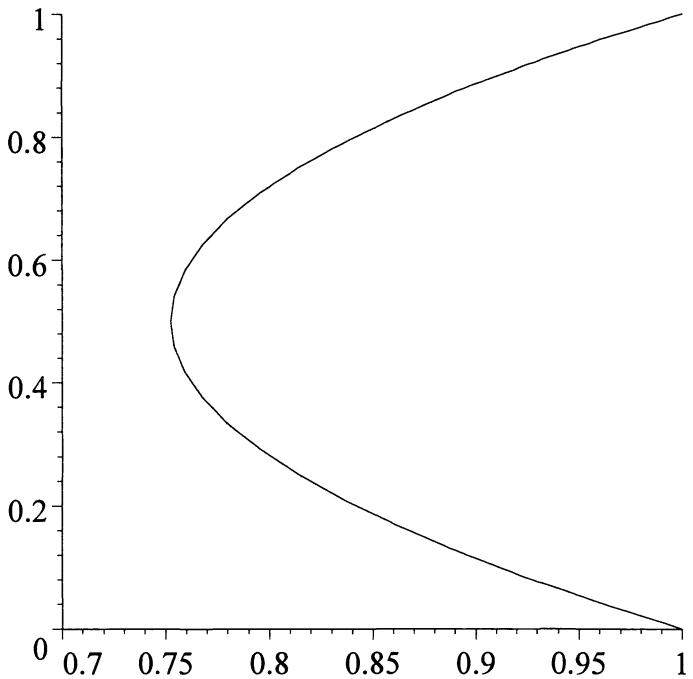
$$(\frac{1}{2}(1-\sqrt{1-\frac{4(\mu_i+\gamma_i)}{\gamma_i}(1-\Re_{0i})}),\frac{1}{2}(1+\sqrt{1-\frac{4(\mu_i+\gamma_i)}{\gamma_i}(1-\Re_{0i})}))$$

coexisting with the locally asymptotically stable disease free equilibrium at $(1,0)$.

The proof of Theorem 3.1 is in the Appendix. Now, we illustrate numerically this backward bifurcation for System (11). We vary $\Re_{0i}$ while the survival probability γ_i and the variable coefficient μ_i remain fixed.

Example 1: Set the following parameter values in System (11):

$$\mu_i = 0.01 \text{ and } \gamma_i = 0.98.$$



The relationship between the proportion of susceptibles and the basic reproductive number $\Re_{0i}$ in patch i. The horizontal axis, $0.7 \leq \Re_{0i} \leq 1$, and the vertical axis, $0 \leq y_{i\infty} \leq 1$.

The disease free equilibrium $(1, 0)$ is locally stable for all values of the parameter, and in Example 1 it is globally stable whenever $\Re_{0i} < 0.7525$. A backward bifurcation occurs at $\Re_{0i} \approx 0.7525$. When $\Re_{0i} \approx 0.7525$, an unstable endemic equilibrium appears, and for values of $\Re_{0i}$ in the interval $(0.7525, 1)$ the system has 2 endemic equilibria coexisting with the disease free equilibrium (see Figure 1 and Theorem 3.1).

4. Two-Patch S-I-S Epidemic Models With Dispersion

Is dispersal the best strategy from the view point of the virus? Who should disperse, susceptibles or infectives? To study the role of dispersal in disease epidemics, we formulate and analyze an $S-I-S$ epidemic model with dispersal of individuals between two patches. We couple Patch 1 and Patch 2 with a simple exchange of a fixed fraction of the population per generation. For each Patch $i \neq j \in \{1,2\}$, let D_{iS} and D_{iI} be the fraction of the susceptible and infective populations that disperse from Patch i to j, respectively. This leads to the following system of equations for the two-patch disease dynamics:

$$\left.\begin{array}{rcl} S_1(t+1) & = & (1-D_{1S})\widetilde{S}_1(t) + D_{2S}\widetilde{S}_2(t) \\ S_2(t+1) & = & D_{1S}\widetilde{S}_1(t) + (1-D_{2S})\widetilde{S}_2(t) \\ I_1(t+1) & = & (1-D_{1I})\widetilde{I}_1(t) + D_{2I}\widetilde{I}_2(t) \\ I_2(t+1) & = & D_{1I}\widetilde{I}_1(t) + (1-D_{2I})\widetilde{I}_2(t) \end{array}\right\} \quad (12)$$

where $\widetilde{S}_i(t) = f_i(T_i(t)) + \gamma_i G_i(\alpha_i(\frac{I_i(t)}{T_i(t)})\frac{I_i(t)}{T_i(t)})S_i(t) + \gamma_i(1-\sigma_i)I_i(t)$, $\widetilde{I}_i(t) = \gamma_i(1 - G_i(\alpha_i(\frac{I_i(t)}{T_i(t)})\frac{I_i(t)}{T_i(t)}))S_i(t) + \gamma_i\sigma_i I_i(t)$ and $0 \leq D_{iS}, D_{iI} \leq 1$. The dispersion coefficients, D_{iS} and D_{iI}, denote the probability of dispersion by susceptible and infective individuals from Patch i to Patch j, respectively; while γ_i denotes the probability of survival in Patch i.

If Patch 2 is empty and there is no dispersion between the two patches ($D_{1S} = D_{1I} = D_{2S} = D_{2I} = \widetilde{S}_2(t) = \widetilde{I}_2(t) = 0$), then System (12) reduces to the single patch model, System (1). In the absence of dispersal, System (12) models two independent patches. Next, we illustrate the potential role of dispersal on patches with identical local dynamics ($f_1 = f_2$) via simple examples.

4.1. Identical Local Patch Dynamics

It is known that dispersion between patches could alter local dynamics [11, 14, 15, 22]. A disease destined to go extinct in Patch i, local basic reproductive number $\Re_{0i} < 1$, could persist in the full system in the presence of dispersion (Example 2). If dispersion rates between two patches are symmetric then System (12) exhibits the same qualitative dynamics as System (1), a one patch system with no dispersion. The joint dynamics live on the invariant set of identical population sizes

$$M = \{(S_1, S_2, I_1, I_2) \mid S_1 = S_2 \text{ and } I_1 = I_2\}.$$

This result appears to be valid only under the assumption of identical local patch ($f_1 = f_2$), local disease dynamics and identical initial conditions. Asymmetric initial conditions, even in the presence of identical local patch dynamics, can generate multiple attractors. Some initial conditions outside of the invariant set M (out-of-phase populations) give rise to dynamics that live outside of M throughout the entire life-history of the local populations. We collect these results below:

Lemma 1: *In System (12), the set of identical local densities*

$$\{(S_1, S_2, I_1, I_2) \mid S_1 = S_2 \text{ and } I_1 = I_2\}$$

is invariant provided $\gamma_1 = \gamma_2, \sigma_1 = \sigma_2, \alpha_1 = \alpha_2, f_1 = f_2$, $G_1 = G_2$, $D_{1S} = D_{2S}$ and $D_{1I} = D_{2I}$.

To prove Lemma 1, note that $S_1(t) = S_2(t)$ and $I_1(t) = I_2(t)$ implies that $\widetilde{S}_1(t) = \widetilde{S}_2(t)$ and $\widetilde{I}_1(t) = \widetilde{I}_2(t)$ whenever $\gamma_1 = \gamma_2, \sigma_1 = \sigma_2, \alpha_1 = \alpha_2, f_1 = f_2$ and $G_1 = G_2$. Consequently, $(1 - D_{1S})\widetilde{S}_1(t) + D_{2S}\widetilde{S}_2(t) = S_1(t+1) = S_2(t+1)$, and

$(1-D_{1I})\widetilde{I}_1(t)+D_{2I}\widetilde{I}_2(t) = I_1(t+1) = I_2(t+1)$. Therefore, $\{(S_1, S_2, I_1, I_2) \mid S_1 = S_2$ and $I_1 = I_2\}$ is an invariant set.

5. Disease-Enhanced Versus Disease-Suppressed Dispersion

Do disease-induced changes in behavior improve the chances of disease persistence? An extreme case of disease-enhanced dispersal is modeled using System (12) with $D_{iS} = 0$, $D_{iI} > 0$. Susceptible individuals are confined to a patch while the infectives are allowed to disperse between patches. Disease-suppressed dispersal occurs in System (12) whenever $D_{iS} > D_{iI}$ while disease-enhanced dispersal occurs when $D_{iI} > D_{iS}$. If $D_{iI} = 0$ while $D_{iS} > 0$ then susceptible individuals disperse between the two patches while the infectives are confined to a patch. One-way disease-suppressed dispersion from Patch 1 to Patch 2 leads to the following system:

$$\left.\begin{array}{rcl} x_1(t+1) &=& (1-D_{1I})\widetilde{x}_1(t) \\ x_2(t+1) &=& D_{1I}\widetilde{x}_1(t) + \widetilde{x}_2(t)) \\ y_1(t+1) &=& \widetilde{y}_1(t) \\ y_2(t+1) &=& \widetilde{y}_2(t) \end{array}\right\} \quad (13)$$

where $\widetilde{x}_i(t) = \frac{\mu_i}{\mu_i+\gamma_i} + \frac{\gamma_i}{\mu_i+\gamma_i}x_i(t)(1-y_i(t)^2) + \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)(1-\sigma_i)$, $\widetilde{y}_i(t) = \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)^2x_i(t) + \frac{\gamma_i}{\mu_i+\gamma_i}\sigma_i y_i(t)$ and $0 < D_{1I} < 1$.

Whenever $D_{1I} = 0$, System (13) reduces to System (11). System (13) has a locally asymptotically stable disease-free equilibrium point at

$$(\frac{(1-D_{1I})\mu_1}{\mu_1+\gamma_1 D_{1I}}, (\frac{D_{1I}\mu_1}{\mu_1+\gamma_1 D_{1I}} + \frac{\mu_2}{\mu_2+\gamma_2})\frac{\mu_2+\gamma_2}{\mu_2}, 0, 0),$$

and some positive initial population sizes lead to the extinction of the disease in

the two patches. In fact, if

$$D_{1I} > 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} \text{ or } D_{1I} < \frac{(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}{2(1-q_1\sigma_1)+(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}$$

then System (13), where $p_i = \frac{\mu_i}{\mu_i+\gamma_i}$ and $q_i = \frac{\gamma_i}{\mu_i+\gamma_i}$ for each $i \in \{1,2\}$, has no endemic equilibrium population size. If

$$D_{1I} = 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} = \frac{(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}{2(1-q_1\sigma_1)+(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}$$

then System (13) has a unique unstable endemic equilibrium population size at the point

$$(\frac{2(1-q_1\sigma_1)}{q_1}, \frac{2(1-D_{1I})p_2q_1(1-q_2\sigma_2)}{q_2((1-D_{1I})p_2q_1+2D_{1I}(1-q_1\sigma_1))}, \frac{1}{2}, \frac{(1-D_{1I})p_2q_1+2D_{1I}(1-q_1\sigma_1)}{2(1-D_{1I})p_2q_1}).$$

Furthermore, the relationships

$$D_{1I} < 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} \text{ and } D_{1I} > \frac{(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}{2(1-q_1\sigma_1)+(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2)q_1}$$

imply that System (13) has two endemic equilibria. Hence, disease persistence depends on initial conditions. Consequently, System (13) is capable of supporting two endemic equilibria coexisting with the locally asymptotically stable disease-free equilibrium (backward bifurcation), with or without dispersion between patches.

The assumption of exclusive disease-driven dispersal, reduces System (12) to the following system of equations:

$$\left.\begin{array}{rcl} x_1(t+1) &=& \widetilde{x}_1(t) \\ x_2(t+1) &=& \widetilde{x}_2(t) \\ y_1(t+1) &=& (1-D_{1I})\widetilde{y}_1(t)+D_{2I}\widetilde{y}_2(t) \\ y_2(t+1) &=& D_{1I}\widetilde{y}_1(t)+(1-D_{2I})\widetilde{y}_2(t) \end{array}\right\} (14)$$

where $\widetilde{x}_i(t) = \frac{\mu_i}{\mu_i+\gamma_i} + \frac{\gamma_i}{\mu_i+\gamma_i}x_i(t)(1-y_i(t)^2) + \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)(1-\sigma_i)$ and $\widetilde{y}_i(t) = \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)^2x_i(t) + \frac{\gamma_i}{\mu_i+\gamma_i}\sigma_i y_i(t)$. System (14) reduces to System (11) whenever $D_{1I} = D_{2I} = 0$. System (14), like the corresponding single-patch model, System (11), has a locally asymptotically stable disease-free equilibrium at

$$(1, 1, 0, 0).$$

Some positive initial population sizes lead to the extinction of the disease in Patch 1 and Patch 2 regardless of the parameter values. System (14), like System (13), generates backward bifurcations with or without dispersion.

Disease persistence in multi-patch systems with dispersal between patches is possible even in situations where the disease is on the brink of extinction without dispersal. To illustrate this possibility we consider the disease-suppressed dispersal model, System (13). It has two fixed points with infectives in Patch 2 and no infectives in Patch 1 at the following levels:

$$(\frac{(1-D_{1I})\mu_1}{\mu_1+\gamma_1 D_{1I}}, \frac{\mu_2+\gamma_2}{2\mu_2}(B \pm \sqrt{(B^2-C)}), 0, \frac{2\mu_2(1-q_2\sigma_2)}{(\mu_2+\gamma_2)q_2(B \pm \sqrt{(B^2-C)})}),$$

where $B = \frac{D_{1I}\mu_1}{\mu_1+\gamma_1 D_{1I}} + \frac{\mu_2}{\mu_2+\gamma_2}, C = \frac{4\mu_2^2(\mu_2+\gamma_2(1-\sigma_2)}{\gamma_2(\mu_2+\gamma_2)}$, $B > \sqrt{C}$and $q_2 = \frac{\gamma_2}{\mu_2+\gamma_2}$. Our simulations illustrate these possibilities:

Example 2: In System (13), let

$$\mu_1 = \mu_2 = 0.3, \gamma_1 = \gamma_2 = 0.9, \sigma_1 = \sigma_2 = 0.9,$$

the local basic reproduction number in each Patch $i \in \{1, 2\}$ is

$$\Re_{0i} = \frac{\gamma_i\sigma_i}{(1-\Re_{id})\gamma_i + \Re_{id}} = 0.675 < 1 - \frac{\gamma_i}{4(\mu_i+\gamma_i)} = 0.8125.$$

Theorem 3.1 implies that, regardless of initial conditions, the disease-free equilibrium is globally asymptotically stable and the disease dies out on each patch if there is no dispersion. Adding dispersal of susceptibles from Patch 1 to Patch 2 alters the outcome. In fact, depending on initial conditions, the disease persists in Patch 2 with dispersal coefficient $DI \geq 0.11$ where there is no disease without dispersal. Example 2 with $DI = 0.5$ shows a system with a stable equilibrium point at $(0.2, 0.286, 0, 1.514)$ and disease persistence in Patch 2.

6. Conclusion

The study of the impact of disease and dispersal on life-history evolution has received little attention. The focus has often been on dispersal [7, 11, 14, 15, 22] or disease [1-3, 5, 8-10, 12, 16, 17, 21]. Here, we have focused on the joint impact of dispersal and disease on the life-history evolution of populations with potentially complex population dynamics. Hence, we have naturally focused on the dynamics of populations with discrete generations and highly nonlinear birth or recruitment functions.

First, we modeled a simple epidemic process on populations with rich and highly complex population dynamics. An S-I-S epidemic process was built on the life-history of a population under a highly non-linear intraspecific competition regime. Earlier work illustrated the possibility of having a population $T(t) = S(t) + I(t)$ living on a two cycle while $I(t)$, infected individuals, remained at the same level [6, 10]. Hence, in the presence of a non-fatal disease, it has been established that $T - dynamics$ can differ from $I - dynamics$ in a single patch. This result is unlikely to be atypical since it is possible to increase the level of

complexity of an epidemic process within a single patch (backward bifurcation and multiple endemic equilibria) even when the $T - dynamics$ is at a globally stable fixed point. We illustrated this result with a simple model (see P. van den Driessche and P. Watmough for the analogue and more general continuous-time versions [29]). Hence, disease can have some impact on local life-history evolution even when it is non-fatal. Many questions remain on the impact of fatal and non-fatal diseases on life-history evolution on a single patch (see [4, 18]). The addition of dispersion adds dynamical richness and, consequently, life-history diversity. Dispersion makes it possible to support multiple attractors and promotes disease persistence.

Disease-induced and disease-suppressed dispersal appear to play a critical role on the generation and support of multiple attractors and in the process increase the likelihood of disease persistence. In other words, dispersion is key to diversity.

7. Appendix

Proof of Theorem 2.1:

The reproduction function for the infected individuals of System (3) is given by

$$h_i(I) = \gamma_i(1 - G_i(\alpha_i(\frac{I}{T_{i\infty}})\frac{I}{T_{i\infty}}))(T_{i\infty} - I) + \gamma_i\sigma_i I,$$

where $h_i : [0, T_{i\infty}] \to [0, T_{i\infty}]$, $h_i(0) = 0$ and $0 \leq I_i(t) = T_{i\infty} - S_i(t) \leq T_{i\infty}$. The set of iterates of h_i is equivalent to the set of density sequence generated by the

second equation in System (3). Differentiation with respect to I gives

$$\begin{aligned} h_i'(I) &= \gamma_i(-\tfrac{1}{T_{i\infty}}(T_{i\infty}-I)G_i'(\alpha_i(\tfrac{I}{T_{i\infty}})\tfrac{I}{T_{i\infty}})(\alpha_i(\tfrac{I}{T_{i\infty}})+\tfrac{I}{T_{i\infty}}\alpha_i'(\tfrac{I}{T_{i\infty}}))-1+ \\ &\quad G_i(\alpha_i(\tfrac{I}{T_{i\infty}})\tfrac{I}{T_{i\infty}}))) + \gamma_i\sigma_i, \\ h_i''(I) &= \gamma_i[\tfrac{2}{T_{i\infty}}(\alpha_i(\tfrac{I}{T_{i\infty}})+\tfrac{I}{T_{i\infty}}\alpha_i'(\tfrac{I}{T_{i\infty}}))G_i'(\alpha_i(\tfrac{I}{T_{i\infty}})\tfrac{I}{T_{i\infty}})- \\ &\quad (\tfrac{(T_{i\infty}-I)}{T_{i\infty}^2}(\alpha_i(\tfrac{I}{T_{i\infty}})+\tfrac{I}{T_{\infty}}\alpha_i'(\tfrac{I}{T_{i\infty}}))^2G_i''(\alpha_i(\tfrac{I}{T_{i\infty}})\tfrac{I}{T_{i\infty}})- \\ &\quad \tfrac{(T_{i\infty}-I)}{T_{i\infty}^2}(2\alpha_i'(\tfrac{I}{T_{i\infty}})+\tfrac{I}{T_{i\infty}}\alpha_i''(\tfrac{I}{T_{i\infty}}))G_i'(\alpha_i(\tfrac{I}{T_{i\infty}})\tfrac{I}{T_{i\infty}})]. \end{aligned}$$

$\Re_{0i} \leq 1$ implies that $h_i'(0) = -\alpha_i(0)\gamma G_i'(0) + \gamma_i\sigma_i \leq 1$. Therefore, the fixed point $\{0\}$ is locally stable under h_i-iteration. Since $G_i' < 0$, $G_i'' \geq 0, \alpha_i(\frac{I_i}{T_{i\infty}}) + \frac{I_i}{T_{i\infty}}\frac{d\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i} > 0$ and $2\frac{d\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i} + \frac{I_i}{T_{i\infty}}\frac{d^2\alpha_i(\frac{I_i}{T_{i\infty}})}{dI_i^2} \leq 0$ we have that, $h_i''(I) < 0$ for $I \in [0, T_{i\infty}]$. The monotonicity condition on h_i' and the fact that $h_i'(0) \leq 1$ imply that $h_i'(I) < 1$ or $h_i(I) < I$ for $I \in (0, T_{i\infty}]$. Hence, $\{I(t)\}_{t\geq 0}$, a strictly decreasing sequence bounded below by zero, converges to the only fixed point of h_i in the interval $[0, T_{i\infty}]$, zero. This proves (a).

$\Re_{0i} > 1$ implies that $h_i'(0) = -\alpha_i(0)\gamma_i G_i'(0) + \gamma_i\sigma_i > 1$ and, therefore, the fixed point $\{0\}$ is locally unstable under h_i-iteration. Let $\overline{I}_i$ denote the smallest positive fixed point of h_i in $[0, T_{i\infty}]$, and note that $h_i(T_{i\infty}) = \gamma_i\sigma_i T_{i\infty} < T_{i\infty}$. The Intermediate Value Theorem guarantees the existence of the positive fixed point $\overline{I}_i \in (0, T_{i\infty})$ satisfying $h_i(\overline{I}_i) = \overline{I}_i$ and $h_i(I) > I$ for $I \in (0, \overline{I}_i)$ and, consequently, $h_i'(\overline{I}_i) \leq 1$. Since $h_i''(I) < 0$ implies that $h_i'(I) < h_i'(\overline{I}_i) \leq 1$ for $I \in (\overline{I}_i, T_{i\infty})$, then $\int_{\overline{I}_i}^{I} h_i'(x)dx < \int_{\overline{I}_i}^{I} dx$ and, we have $h_i(I) < I$ for $I > \overline{I}_i$. Hence, h_i has a unique positive fixed point $\overline{I}_i \in (0, T_{i\infty})$. Furthermore, $h_i(I) > I$ for $I \in (0, \overline{I}_i)$ and $h_i(I) < I$ for $I \in (\overline{I}_i, T_{i\infty}]$.

To establish the global stability of $\overline{I}_i$, we first prove the nonexistence of non-trivial two-cycles for h_i. Note that $1 + h_i'(I) = 1 + \gamma_i(-\frac{\alpha_i}{T_{i\infty}}(T_{i\infty} - I)G_i'(\frac{\alpha_i I}{T_{i\infty}}) - 1 + G_i(\frac{\alpha_i I}{T_{i\infty}})) + \gamma_i\sigma_i \geq 1 - \gamma_i + \gamma_i\sigma_i > 0$. Hence, $1 + h_i'(I) \neq 0$ for $I \in [0, T_{i\infty}]$,

that is, h_i has no non-trivial 2-cycles. Suppose h_i has a non-trivial 2-cycle $\{p, q\}$ where $p, q \in [0, T_{i\infty}]$, then $h_i(p) = q$ and $h_i(q) = p$ where $p \neq q$. The Mean Value Theorem guarantees the existence of a point r between p and q such that $h_i'(r) = \frac{h_i(p)-h_i(q)}{p-q} = -1$, and $1 + h_i'(r) = 0$, a contradiction. Hence, h_i has no non-trivial 2-cycles in $[0, T_{i\infty}]$. Sharkovskii's Theorem and $1 + h_i'(I) \neq 0$ imply the nonexistence of any $m - cycles$ for $m > 1$. From a result of Cull [13], the nonexistence of non-trivial 2-cycles for h_i implies global stability of the positive fixed point $\overline{I}_i$.

Proof of Theorem 2.3:

Recall that, $T_i(t + 1) = (\mu_i + \gamma_i)T_i(t)$ and $\Re_{id} = \frac{\mu_i}{1-\gamma_i}$. Hence, $\Re_{id} > 1$ implies T_i increases geometrically and $\Re_{id} < 1$ implies T_i decreases geometrically. To establish the result, we prove that if $\Re_{0i} \leq 1$ then the solutions $(x_i(t), y_i(t))$ of System (8) approach the equilibrium $(1, 0)$, as $t \to \infty$. If $\Re_{0i} > 1$, we proceed exactly as in the proof of Theorem 2.1 to prove that the solutions $(x_i(t), y_i(t))$ of System (8) approach a unique positive endemic equilibrium, $(\overline{x}_i, \overline{y}_i) \in (0, \infty) \times (0, \infty)$, as $t \to \infty$.

For the proof of Theorem 2.3, the reproduction function for the infected individuals of System (8) is

$$h_i(y_i) = \frac{\gamma_i}{\mu_i + \gamma_i}(1 - G_i(\alpha_i(y_i)y_i))(1 - y_i) + \frac{\gamma_i}{\mu_i + \gamma_i}\sigma_i y_i$$

where $h_i : [0, 1] \to [0, 1]$. Now, we proceed exactly as in the proof of Theorem 2.1 to establish the result.

Proof of Theorem 3.1:

The reproduction function for the proportion of infected individuals of System

(11) is

$$h_i(y_i) = \frac{\gamma_i}{\mu_i + \gamma_i} y_i^2(1 - y_i) + \frac{\gamma_i}{\mu_i + \gamma_i} \sigma_i y_i$$

where $h_i : [0, 1] \to [0, 1]$.

Notice that $\{0\}$ is a locally asymptotically stable fixed point of h_i for all values of the parameters. To prove (a), note that $0 < \Re_{0i} < 1 - \frac{\gamma_i}{4(\mu_i+\gamma_i)}$ implies that h_i has no other fixed points in the interval $(0, 1]$.Consequently, $h_i(y_i) < y_i$ for each $y_i \in (0, 1]$, and the fixed point $\{0\}$ is globally stable under h_i iterations. Hence, the disease free equilibrium point $(1, 0)$ is globally stable in System (11). $\Re_{0i} = 1 - \frac{\gamma_i}{4(\mu_i+\gamma_i)}$ implies that the fixed points of h_i are $\{0\}$ and $\{\frac{1}{2}\}$. Since $\{0\}$ is locally asymptotically stable, the result is immediate.

To prove (c) notice that $\Re_{0i} > 1 - \frac{\gamma_i}{4(\mu_i+\gamma_i)}$ implies that the fixed points of h_i are $\{0\}$, $\{\frac{1}{2}(1-\sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1 - \Re_{0i})})\}$ and $\{\frac{1}{2}(1+\sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1 - \Re_{0i})})\}$. Since $\{0\}$ is locally asymptotically stable, the fixed point $\{\frac{1}{2}(1-\sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1 - \Re_{0i})})\}$ is unstable and $\{\frac{1}{2}(1 + \sqrt{1 - \frac{4(\mu_i+\gamma_i)}{\gamma_i}(1 - \Re_{0i})})\}$ is locally asymptotically stable. This establishes Theorem 3.1.

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